In the claims:

Please cancel claims 3-4, 7-9, and 11-13 without prejudice or disclaimer to applicants' right to pursue the subject matter of this claim in a later filed application. Please also amend claims 1-2, 5 and 14 under the provisions of 37 C.F.R. §1.121(c) as follows. A marked up version of amended claim 4 wherein the deleted material is in brackets and the inserted material is underlined is attached hereto as **Exhibit B**:

--1. (Amended) A transgenic mouse whose genome comprise a recombinant DNA sequence comprising a nerve tissue specific promoter operatively linked to a DNA sequence encoding amyloid-beta peptide alcohol dehydrogenase (ABAD), wherein said transgenic mouse exhibits elevated levels of basal amyloid precursor protein (APP).--

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--2. (Amended) The transgenic mouse of claim 1, wherein the promoter is platelet derived growth factor (PDGF)-B-chain promoter.--

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--5. (Amended) A method for evaluating in a transgenic mouse the potential therapeutic effect of a compound for treating Alzheimer's disease-like pathology in a human, which comprises:

(a) administering the compound to a transgenic mouse whose genome comprise a recombinant DNA sequence comprising a nerve tissue specific promoter operatively linked to a DNA sequence which encodes amyloid-beta peptide alcohol dehydrogenase (ABAD), and

(b) determining the therapeutic effect of the compound on the transgenic mouse by monitoring basal synaptic transmission or synaptic plasticity or basal levels of ATP, wherein an increase in basal synaptic transmission or synaptic plasticity or basal levels of ATP indicates that the compound would have a potential therapeutic effect on Alzheimer's disease-like pathology in a human.--

Q16

--14.(Amended) A transgenic mouse whose genome comprise a recombinant DNA sequence comprising:

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(a) a nerve tissue specific promoter; and

(b) a DNA sequence which encodes amyloid-beta peptide alcohol dehydrogenase (ABAD),

wherein the promoter and the DNA sequence which

encodes amyloid-beta peptide alcohol dehyrdogenase are operatively linked to each other and integrated in the genome of the mouse, and

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wherein said mouse exhibits at least one phenotype from the group consisting of: overexpression of ABAD in brain, increased ATP level in cerebral cortex; decreased lactate level in cerebral cortex and lower beta-hydroxybutyrate levels in cerebral cortex which has been subjected to cerebral ischemia.--

REMARKS

Claims 1-16 were pending in the subject application. Applicants have hereinabove canceled claims 3-4, 7-9 and 11-13 without prejudice or disclaimer to applicants right to pursue the subject matter of these claims in a later-filed application and amended claims 1-2, 5 and 14. Support for these amendments may be found inter alia in the specification as follows: Claim 1: page 8, lines 3-9; Claim 2: page 8, lines 28-30; Claim 5: page 8, lines 11-24; and Claim 14: page 9, lines 3-14. Claims 1-2, 5 and 14 do not involve any issue of new matter. Therefore, entry of this amendment is respectfully requested such that claims 1-2, 5-6, 10 and 14-16 will be pending.

REMARKS

Abstract: